Unfortunately, the new paper [4] does not provide data on wing kinematics, but it is reasonable to think that the wing motion of hovering bats and moths is similar. Indeed, the new images of the bat wing and its leading edge vortex are uncannily similar to images captured previously for moths (Figure 1) [18,19]. As elegantly described by the great neuroethologist Ken Roeder [20], bats and moths are engaged in a deadly evolutionary arms race for command of the night sky. It is intriguing to note that these creatures are nevertheless united by the laws of physics.

References

1. Wang, X., Kellner, A., Zhou, Z., and de Almeida Campos, D. (2005). Pterosaur diversity and faunal turnover in Cretaceous terrestrial ecosystems in China. Nature *437*, 875–879.
2. Dickinson, M.H. (2006). Insect flight. Curr. Biol. *16*, R309–R314.
3. Kukalova-Peck, J. (1992). The "Uniramia" do not exist: the ground plan of the Pterygota as revealed by Permian Diaphanopterodea from Russia (Insecta: Paleodicyteroidea). Can. J. Zool. *70*, 236–255.
4. Muijres, F.T., Johansson, L.C., Barfield, R., Wolf, M., Spedding, G.R., and Hedenstrom, A. (2008). Leading-edge vortex improves lift in slow-flying bats. Science *319*, 1250–1253.
5. Videler, J. (2006). Avian Flight (Oxford: Oxford University Press).
6. Ellington, C.P. (1984). The aerodynamics of hovering insect flight. I. The quasi-steady analysis. Phil. Trans. R. Soc. Lond. B *305*, 1–15.
7. Ellington, C.P. (1984). The aerodynamics of insect flight. IV. Aerodynamic mechanisms. Phil. Trans. R. Soc. Lond. B *305*, 79–113.
8. Maxworthy, T. (1979). Experiments on the Weis-Fogh mechanism of lift generation by insects in hovering flight Part 1. Dynamics of the 'fling'. J. Fluid Mech. *93*, 47–63.
9. Weis-Fogh, T. (1973). Quick estimates of flight fitness in hovering animals, including novel mechanisms for lift production. J. Exp. Biol. *59*, 169–230.
10. Dickinson, M.H., and Gotz, K.G. (1993). Unsteady aerodynamic performance of model wings at low Reynolds-numbers. J. Exp. Biol. *174*, 45–64.
11. Dickinson, M.H., Lehmann, F.O., and Sane, S.P. (1999). Wing rotation and the aerodynamic basis of insect flight. Science *284*, 1954–1960.
12. Ellington, C.P., van den Berg, C., Willmott, A.P., and Thomas, A.L.R. (1996). Leading-edge vortices in insect flight. Nature *384*, 626–630.
13. Swartz, S.M., Bishop, K.L., and Ismael-Aguirre, M.F. (2006). Bat flight aerodynamics: new insights from three-dimensional kinematic analysis. In Functional and Evolutionary Ecology of Bats, T.H. Kunz, A. Zubaid, and G.F. McCracken, eds. (Oxford: Oxford University Press).
14. Raffel, M., Willert, C., and Kompenhans, J. (1998). Particle Image Velocimetry: A Practical Guide (Heidelberg: Springer-Verlag).
15. Milne-Thomson, L.M. (1966). Theoretical Aerodynamics, 4th Edition (New York: Macmillan).
16. Warrick, D., Tobalske, B.W., and Powers, D.R. (2005). Aerodynamics of the hovering hummingbird. Nature *435*, 1094–1097.
17. Videler, J.J., Stamhuis, E.J., and Povel, G.D.E. (2004). Leading-edge vortex lifts swifts. Science *306*, 1960–1962.
18. Willmott, A.P., Ellington, C.P., and Thomas, A.L.R. (1997). Flow visualization and unsteady aerodynamics in the flight of the hawkmoth *Manduca sexta*. Phil. Trans. R. Soc. Lond. B *352*, 303–316.
19. Bomphrey, R.J., Lawson, N.J., Harding, N.J., Taylor, G.K., and Thomas, A.L.R. (2005). The aerodynamics of *Manduca sexta*: digital particle image velocimetry analysis of the leading-edge vortex. J. Exp. Biol. *208*, 1079–1094.
20. Roeder, K.D. (1963). Nerve Cells and Insect Behavior (Cambridge: Harvard University Press).

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Cell Shape: Taking the Heat

Preservation of cell architecture under physically stressful conditions is a basic requirement for many biological processes and is critical for mechanosensory systems built to translate subtle changes in cell shape into changes in organism behaviour. A new study reveals how an extracellular protein — Spam — helps mechanosensory organs in the fruit fly to withstand the effects of the water loss that accompanies heat shock.

Patricia Kunda, Jennifer L. Rohn and Buzz Baum

Sensory systems allow animals to detect minute changes in their environment, including those accompanying adverse conditions. This information can then be used to induce an appropriate response, for example to guide the direction of movement or the selection of an adequate place to lay eggs. But some potentially harmful environmental changes, such as the daily fluctuations in ambient temperature, cannot be easily avoided; instead, they must be tolerated. This problem is likely to be particularly acute for sensory systems themselves, as they have evolved to be optimised for sensitivity to environmental changes.

Exposing a small animal such as a fruit fly to a simple heat shock will compromise the ability of its sensory systems to function by altering body temperature and the rates of many of the biochemical events involved in signalling. In addition, dry heat can induce a loss of water, resulting in osmotic shock. As mechanosensory organs are constructed to translate mechanical events at the cellular level into changes in organismal behaviour, this latter problem is likely to be especially acute. Under normal conditions, the movement of a bristle on the back of the fly will induce changes in the lipid bilayer and the underlying cortical cytoskeleton of mechanosensitive neurons, altering the regulated flow of ions across the plasma membrane through transient receptor potential (TRP) channels [1], in turn triggering an action potential that passes to the central ganglion to ultimately alter fly behaviour [2–4]. By disturbing the shape of the cells involved in mechanotransduction, osmotic shock is likely to compromise performance and to damage the delicate machinery involved in sensing bristle movement. Moreover, since TRP channels function in both mechanosensation and osmoregulation, osmotic shock may also induce aberrant signalling. How mechanosensory systems cope with such everyday environmental changes is therefore a pressing question in biology.

A recent paper from Charles Zuker and colleagues published in *Nature* sheds light on this problem [5]. By screening for changes in fruit fly behaviour, these authors identified a normally viable mutation in the gene *spacemaker (spam)* that sensitized flies to the effects of a 37°C heat shock. Whereas wild-type flies appeared unaffected by this treatment, mutant animals manifested severe and irreversible defects in their ability to walk, feed and fly. Intriguingly, Spam is a secreted protein containing several epidermal growth factor and laminin G-like repeats and was recently identified by the Zuker lab to be a structural component of the *Drosophila* eye [6]. In the eye, Spam binds to Prominin, a conserved integral membrane protein [7], to generate an intra-rhabdomeric space which prevents rhabdomeres from touching each other, thereby improving specific aspects of ommatidial function [8]. Given its specific role in eye morphogenesis, what might Spam be doing in the mechanosensory system to enable it to resist thermal stress?

Cook *et al.* [5] used a combination of neurophysiological and cell biological methods to elucidate a possible mechanism for Spam's action. First, to test the functionality of mechanoreceptors in the *spam* mutant, they performed electrophysiological recordings from two structures: bristle mechanoreceptors and antennal chordotonal organs. While control and *spam* mutant flies had a normal response to stimulation at permissive temperatures, a 30-minute exposure at 37°C irrevocably blocked the ability of *spam* mutant flies to respond to mechanical stimulation. Using electron microscopy to examine the cells involved, the authors observed a dramatic change in the morphology of the mechanoreceptor organ in mutant flies exposed to elevated temperatures. In this case, the integrity of cells in the system was clearly compromised because cytoplasm was seen to invade the extracellular space, which, like the inter-rhabdomeral space in the eye, makes up a significant part of the organ.

In exploring how a simple change in temperature could have such unexpected effects on cellular organisation in *spam* mutant flies, Cook *et al.* [5] looked at the effects of the heat-shock regimen on water loss. Both wild-type and mutant flies were found to lose 25% of their water content through evaporation as a consequence of this treatment [3]. Importantly, the researchers were able to prove that this water loss and the consequent change in hemolymph osmolarity were responsible for the observed defects in *spam* mutant flies by demonstrating that these flies do tolerate a heat shock in conditions of high humidity and by inducing similar defects by injecting flies with hypertonic fluid.

This discovery provided Cook *et al.* [5] with a simple way to test whether Spam plays a direct role in protecting cells from the effects of osmotically induced mechanical stress. To do so, they turned to fly cell culture. Exploiting the fact that extracellular Spam becomes bound and anchored at the surface of Prominin-expressing cells [6], they co-expressed Prominin and Spam in cells that do not normally express either protein to induce the formation of an extracellular Spam coat, visualised using Spam antibodies. They then evaluated how this extracellular Spam coat affected the ability of cells to withstand both hypertonic and hypotonic shock. Remarkably, Spam-decorated cells appeared relatively unaffected by fluctuations in osmolarity, while neighbouring uncoated cells suffered huge changes in shape and size. The authors suggest that the effects of Spam in cell culture might mimic the action of extracellular Spam in the mechanosensory system. To exclude the involvement of the actin cytoskeleton in the process, they treated cells with actin poisons. Once again, the co-expression of Spam and Prominin enabled cells to maintain their shape, while control cells were rapidly deformed by microtubule-based extensions following the loss of actin filaments [9]. Under these conditions, Spam-coated cells were also found to be significantly stiffer than control cells. These experiments suggest a simple mechanism by which Spam, acting in an analogous fashion to the cell wall of yeast and bacteria, provides cells with a mechanical defense against cell-shape changes induced by hypo-osmotic shock [10]. It is not clear how this coat protects cells under hypertonic conditions, but it is conceivable that extracellular Spam also acts as an osmotic buffer in a way similar to that proposed for the extracellular matrix in other systems [11].

Proving that this *in vitro* system is a good model for the action of extracellular Spam within the mechanosensory system and identifying the functional partners that act together with Spam in the context of the mechanosensory system will be important goals for future research. In an *in vivo* context, we also need to understand how a Spam coat can protect cells from unpredictable changes in the environment without inhibiting the normal physiological function of the cells involved. Given that Spam localizes to specific substructures within the mechanosensory organ, this may be achieved by confining the accumulation of extracellular Spam to weak points in the system, namely junctions between mechanically linked structures, leaving the rest of the system free to function. If this is the case, it is clear that cells of the mechanosensory organ will need to employ a host of additional strategies to function normally in the face of dramatic changes in osmotic pressure. Such strategies may include the regulated flux of water and ions to regulate osmotic potential, the accumulation of intracellular osmostabilants that act like trehalose in yeast to buffer cells against changes in extracellular osmotic pressure [12], and systems to mechanically stabilize the membrane, such as annexins [13] and changes in the organization of the supporting actin cytoskeleton [14,15].

Importantly, this study demonstrates the need for specific mechanisms to ensure that normal cell physiology continues to function robustly when faced with changes in environmental conditions. This places this work in the context of recent studies which identified a role for the extracellular portion of Crumbs, a protein with domains similar to those in Spam, in the protection of the fly eye from progressive light-induced degeneration [7]. Prominin has also been implicated in human retinal degeneration [6], raising the possibility that Spam, Prominin and Crumbs also act in broadly similar ways to limit environmentally induced damage in the eye. Such robust biological systems in nature serve as an historical record of the challenges faced by organisms during their evolution. A better understanding of the complex mechanisms underlying sensory input, behavioural output resistance and resilience to external changes will also prove invaluable as dramatic ecological changes and climate fluctuations increase in the future.

References

1. Clapham, D.E. (2003). TRP channels as cellular sensors. Nature *426*, 517–524.
2. Sukharev, S., and Corey, D.P. (2004). Mechanosensitive channels: multiplicity of families and gating paradigms. Sci. STKE *2004*, re4.
3. Walker, R.G., Willingham, A.T., and Zuker, C.S. (2000). A Drosophila mechanosensory transduction channel. Science *287*, 2229–2234.
4. Christensen, A.P., and Corey, D.P. (2007). TRP channels in mechanosensation: direct or indirect activation? Nat. Rev. Neurosci. *8*, 510–521.
5. Cook, B., Hardy, R.W., McConnaughey, W.B., and Zuker, C.S. (2008). Preserving cell shape under environmental stress. Nature *452*, 361–364.

6. Zelhof, A.C., Hardy, R.W., Becker, A., and Zuker, C.S. (2006). Transforming the architecture of compound eyes. Nature *443*, 696–699.
7. Johnson, K., Grawe, F., Grzeschik, N., and Knust, E. (2002). Drosophila crumbs is required to inhibit light-induced photoreceptor degeneration. Curr. Biol. *12*, 1675–1680.
8. Osorio, D. (2007). Spam and the evolution of the fly's eye. BioEssays *29*, 111–115.
9. Kiger, A.A., Baum, B., Jones, S., Jones, M.R., Coulson, A., Echeverri, C., and Perrimon, N. (2003). A functional genomic analysis of cell morphology using RNA interference. J. Biol. *2*, 27.
10. Hohmann, S., Krantz, M., and Nordlander, B. (2007). Yeast osmoregulation. Methods Enzymol. *428*, 29–45.
11. Lu, Y., Parker, K.H., and Wang, W. (2006). Effects of osmotic pressure in the extracellular matrix on tissue deformation. Philos. Transact. A Math. Phys. Eng. Sci. *364*, 1407–1422.
12. Felix, C.F., Moreira, C.C., Oliveira, M.S., Sola-Penna, M., Meyer-Fernandes, J.R., Scofano, H.M., and Ferreira-Pereira, A. (1999). Protection against thermal denaturation by trehalose on the plasma membrane H+-ATPase from yeast. Synergetic effect between trehalose and phospholipid environment. Eur. J. Biochem. *266*, 660–664.
13. Hill, W.G., Kaetzel, M.A., Kishore, B.K., Dedman, J.R., and Zeidel, M.L. (2003). Annexin A4 reduces water and proton permeability of model membranes but does not alter aquaporin 2-mediated water transport in isolated endosomes. J. Gen. Physiol. *121*, 413–425.
14. Rasmussen, M., Alexander, R.T., Darborg, B.V., Mobjerg, N., Hoffmann, E.K., Kapus, A., and Pedersen, S.F. (2008). Osmotic cell shrinkage activates ezrin/radixin/moesin (ERM) proteins: activation mechanisms and physiological implications. Am. J. Physiol. Cell. Physiol. *294*, C197–C212.
15. Kunda, P., Pelling, A.E., Liu, T., and Baum, B. (2008). Moesin controls cortical rigidity, cell rounding, and spindle morphogenesis during mitosis. Curr. Biol. *18*, 91–101.

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Insect Migration: Do Migrant Moths Know Where They Are Heading?

Moth migration has been assumed to involve hitching a ride in favorable winds. A new study has shown that silver Y moths migrate only on nights when winds would displace them southward, implying that they detect their direction of movement while airborne, likely by a magnetic sense.

Ring T. Cardé

Migration is a persistent, straightened-out movement that takes an organism from a habitat lacking a resource to another, more favorable location. During migration, the organism's 'vegetative' behaviors such as feeding and reproduction are temporarily suppressed [1]. In insects, the distances traveled can exceed 3,000 km, as in the case of monarch butterflies, *Danaus plexippus*, flying in autumn from New England to their overwintering home in the Sierra Madre Mountains in Mexico. This butterfly migrates in daytime, and usually near ground level. Using a novel tethering system to simulate free flight, the monarch's only orientation mechanism verified so far is a sun compass; earlier evidence that the monarch uses magnetic cues or polarized light in navigation has been discounted [2,3]. Migration can also be a relatively local phenomenon, on the order of a kilometer or less, as with the seasonal movement of the black bean aphid, *Aphis fabae*, which has two generations on a woody host before a springtime flight that takes it into bean fields where it reproduces asexually until fall, when sexual, winged forms return to the woody host [4].

Migration thus need not involve return trips by the same individuals, nor does a migrant need to direct its track [4], as the monarch does; in small insects, like aphids, migration is subject to the vagaries of the prevailing wind. While much debate remains on the proximate mechanisms governing these mass movements, the ultimate selective force explaining these displacements is spread of reproductive efforts in time and space [5].

Many noctuid moths migrate in spring from mild-weather temperate regions to exploit higher latitude regions in summer. Migration to high latitudes has been likened to a Pied Piper effect, leading migrants to exploit a temporarily favorable environment, but without the prospect of their progeny surviving the winter [6]. The question of whether the descendants of wind-borne migrant moths have a return migration in autumn to lower latitudes has been debated, but there is now evidence in some species from mark-release-recapture experiments — in which many moths are marked and a very few are recovered at considerable distances from their release point — that such to and fro migration can occur (for example [7]). Such migrants have been assumed to hitch a ride in both directions on winds that are usually seasonally 'favorable'.

The silver Y moth, *Autographa gamma* (Figure 1), migrates in autumn from northern Europe to North Africa and the Mediterranean basin; in spring its descendants re-migrate northward. In a recent report in *Current Biology* [8], the vertical-looking radar (VLR) technique [9] was coupled with meteorological data to provide new insights into how this noctuid moth heads toward its overwintering habitat, in this case en route over central England toward the Mediterranean basin. What is unique in this study is the simultaneous measurement by VLR of the body orientation and track directions of numerous individual moths on many evenings and the availability of wind movement data at relevant altitudes.

In autumn, mass migratory flights occur on those nights when the wind flow is favorable for rapid southward movement and moths are concentrated at altitudes that maximize their displacement. VLR pinpoints the body orientation of individual moths as they pass hundreds of meters overhead, and thereby can indicate whether they are contributing to their downwind displacement by heading with the wind. The silver Y does not use changes at ground level in temperature, humidity, wind speed, wind direction or barometric pressure to forecast a directionally favorable wind above — indeed, in autumn the wind direction at migratory height was found to be randomly distributed and nights with a southerly wind flow at migratory